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      Set  Items  Description
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Cost is in DialUnits
? b 410
       30mar02 09:47:02 User208760 Session D2029.1
            $0.33    0.095 DialUnits File1
     $0.33  Estimated cost File1
     $0.33  Estimated cost this search
     $0.33  Estimated total session cost   0.095 DialUnits
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File 410:Chronolog(R)  1981-2002/Feb
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      Set  Items  Description
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HILIGHT set on as ''
? begin 5,73,155,399
       30mar02 09:47:06 User208760 Session D2029.2
            $0.00    0.076 DialUnits File410
     $0.00  Estimated cost File410
     $0.01  TELNET
     $0.01  Estimated cost this search
     $0.34  Estimated total session cost   0.171 DialUnits

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      Set  Items  Description
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Ref   Items  Index-term
E1        2  AU=CO MA
E2       16  AU=CO MAN SUNG
E3        0 *AU=CO MANG?
E4        1  AU=CO MARK A
E5        1  AU=CO MARTI F
E6        2  AU=CO MARY DAWN T
E7        2  AU=CO MC
E8        1  AU=CO MPAGNO A.
E9       29  AU=CO MS
E10       7  AU=CO N
E11       1  AU=CO N.
E12       1  AU=CO N.D.

          Enter P or PAGE for more
? s e1,e2,e9
               2  AU=CO MA
              16  AU=CO MAN SUNG
              29  AU=CO MS
      S1      47  E1,E2,E9
? s s1 and b7?
              47  S1
           19410  B7?
      S2       0  S1 AND B7?
? s (b7(w)1)(5n)(antibod?)0 and (b7(w)2)(5n)(antibod?) and (cancer? or tumor? or
tumour? or leukemia? or lymphoma?)
Processing
Processing
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         8351538  1
               0  ANTIBOD?)0
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              0  B7(W)1(5N)ANTIBOD?)0
          15219  B7
        8099045  2
        1693117  ANTIBOD?
            321  B7(W)2(5N)ANTIBOD?
        1828048  CANCER?
        1859880  TUMOR?
         249841  TUMOUR?
         476950  LEUKEMIA?
         260141  LYMPHOMA?
      S3       0  (B7(W)1)(5N)(ANTIBOD?)0 AND (B7(W)2)(5N)(ANTIBOD?) AND
                  (CANCER? OR TUMOR? OR TUMOUR? OR LEUKEMIA? OR LYMPHOMA?)
? s (b7(w)1)(5n)(antibod?) and (b7(w)2)(5n)(antibod?) and (cancer? or tumor? or
tumour? or leukemia? or lymphoma?)
Processing
Processing
Processing
          15219  B7
        8351538  1
        1693117  ANTIBOD?
            295  B7(W)1(5N)ANTIBOD?
          15219  B7
        8099045  2
        1693117  ANTIBOD?
            321  B7(W)2(5N)ANTIBOD?
        1828048  CANCER?
        1859880  TUMOR?
         249841  TUMOUR?
         476950  LEUKEMIA?
         260141  LYMPHOMA?
      S4      24  (B7(W)1)(5N)(ANTIBOD?) AND (B7(W)2)(5N)(ANTIBOD?) AND
                  (CANCER? OR TUMOR? OR TUMOUR? OR LEUKEMIA? OR LYMPHOMA?)
? rd s4
...completed examining records
      S5      11  RD S4 (unique items)
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 5/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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11958232   BIOSIS NO.: 199900204341
B7-2 expressed on EL4 lymphoma suppresses antitumor immunity by an
  interleukin 4-dependent mechanism.
AUTHOR: Stremmel C; Greenfield E A; Howard E; Freeman G J; Kuchroo V K(a)
AUTHOR ADDRESS: (a)Center for Neurological Diseases, Department of
  Neurology, Brigham and Women's Hospital, Harvard**USA
JOURNAL: Journal of Experimental Medicine  189 (6):p919-930 March 15, 1999
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: For T cells to become functionally activated they require at
  least two signals. The B7 costimulatory molecules B7-1 and B7-2 provide
  the "second signal" pivotal for T cell activation. In this report, we
  studied the relative roles of B7-1 and B7-2 molecules in the induction of
  antitumor immunity to the T cell thymoma, EL4. We generated EL4
  tumor cells that expressed B7-1, B7-2, and B7-1+B7-2 by
  transfecting murine cDNAs. Our results demonstrate that EL4-B7-1 cells
  are completely rejected in syngeneic mice. Unlike EL4-B7-1 cells, we find
  that EL4-B7-2 cells are not rejected but progressively grow in the mice.
  A B7-1- and B7-2-EL4 double transfectant was generated by introducing
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B7-2 cDNA into the EL4-B7-1 **tumor** line that regressed in vivo. The EL4-B7-1+B7-2 double transfectant was not rejected when implanted into syngeneic mice but progressively grew to produce **tumors**. The double transfectant EL4 cells could costimulate T cell proliferation that could be blocked by anti-**B7-1 antibodies**, anti-**B7-2 antibodies**, or hCTLA4 immunoglobulin, showing that the B7-1 and B7-2 molecules expressed on the EL4 cells were functional. In vivo, treatment of mice implanted with double-transfected EL4 cells with anti-**B7-2** monoclonal **antibody** resulted in **tumor** rejection. Furthermore, the EL4-B7-2 and EL4-B7-1+B7-2 cells, but not the wild-type EL4 cells, were rejected in interleukin 4 (IL-4) knockout mice. Our data suggests that B7-2 expressed on some T cell **tumors** inhibits development of antitumor immunity, and IL-4 appears to play a critical role in abrogation of the antitumor immune response.

5/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10984173 BIOSIS NO.: 199799605318
Expression of B7-2 (CD86) molecules by Reed-Sternberg cells of Hodgkin's disease.
AUTHOR: Van Gool S W; Delabie J; Vandenberghe P; Coorevits L; De Wolf-Peeters C; Ceuppens J L(a)
AUTHOR ADDRESS: (a)Lab. Exp. Immunol., Fac. Med., Onderwijs en Navorsing, Herestraat 49, B-3000 Leuven**Belgium
JOURNAL: Leukemia (Basingstoke) 11 (6):p846-851 1997
ISSN: 0887-6924
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Ligation of CD28 on T cells with its natural ligands B7-1 (CD80) or B7-2 (CD86) provides a major costimulatory signal for T cells and is of potential importance for **tumor** rejection. We previously reported a strong expression of B7-1 on Reed-Sternberg cells and anaplastic large cell **lymphoma** cells. We report here our findings on B7-2 expression by malignant **lymphomas** (n = 70). B7-2 was present on the neoplastic cells of anaplastic large cell **lymphoma** in two of three cases studied, and on a subpopulation of the malignant cells in one out of four cases of follicular **lymphoma**. B7-2 was not expressed by the neoplastic cells of the other non-Hodgkin's **lymphomas** (n = 32), including T cell-rich B cell **lymphoma**. In contrast, Reed-Sternberg cells in lymph nodes affected by Hodgkin's disease are strongly positive for B7-2 (n = 31). Evidence for a functional correlate of this expression was obtained by our findings that the combination of anti-**B7-1** and anti-**B7-2** monoclonal **antibodies** was more effective than each separately in blocking allogeneic T cell activation (proliferation and cytokine secretion) by Hodgkin's disease-derived cell lines as stimulators. The possible role of B7-1 and B7-2 expression for the course and symptomatology of Hodgkin's disease is discussed.

5/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10971236 BIOSIS NO.: 199799592381
Cytokine modulation of T-lymphocyte activation by intestinal smooth muscle cells.
AUTHOR: Hogaboam Cory M(a); Snider Denis P; Collins Stephen M
AUTHOR ADDRESS: (a)Dep. Pathol., Univ. Michigan Medical Sch., 1301 Catherine Road, Ann Arbor, MI 48105**USA
JOURNAL: Gastroenterology 112 (6):p1986-1995 1997

ISSN: 0016-5085
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background & Aims: Interleukin 1-beta (IL-beta) and **tumor** necrosis factor alpha (TNF-alpha) are present in the neuromuscular layers during intestinal inflammation and directly affect intestinal smooth muscle function. We investigated whether IL-1-beta and TNF-alpha modulate T-cell activation by murine intestinal smooth muscle cells (ISMCs). Methods: alpha- and gamma- actin expression in ISMCs was confirmed using reverse-transcription polymerase chain reaction (RT-PCR). ISMCs were analyzed for class II major histocompatibility complex (MHC), intercellular adhesion molecule 1 (ICAM-1), and B7 before and after exposure to interferon gamma (IFN-gamma; 100 or 1000 U/mL) in the presence or absence of IL-1-beta (10 ng/mL) or TNF-alpha (5 ng/mL) for 72 hours. T-cell proliferation on cytokine-pretreated ISMCs was measured in the absence or presence of anti-B7 antibodies. Results: In a dose-dependent fashion, IFN-gamma-pretreated ISMCs expressed MHC class II, ICAM-1, and B7-2, and stimulated T-cell proliferation. Pretreatment of ISMCs with IL-1-beta and IFN-gamma reduced MHC class II and ICAM-1 expression and inhibited T-cell proliferation. When added with 100 U/mL IFN-gamma, TNF-alpha enhanced MHC class II and ICAM-1 expression on ISMCs and T-cell proliferation. However, TNF-alpha and 1000 U/mL IFN-gamma significantly decreased MHC class II expression and T-cell proliferation. Anti-**B7**-2 monoclonal **antibody** but not anti-**B7**-**1** inhibited T-cell proliferative responses by gt 50%. Conclusions: Because IL-1-beta, TNF-alpha, and T cells are present in the intestinal muscle layers during inflammation, these cytokines may serve to modulate the activation of T cells in this site.

5/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10945065 BIOSIS NO.: 199799566210
Generation of dendritic cell-like antigen-presenting cells in long-term mixed leucocyte culture: Phenotypic and functional studies.
AUTHOR: Gao J-X; Madrenas J; Zeng W; Zhong R; Grant D(a)
AUTHOR ADDRESS: (a)Dep. Surgery, Univ. Hospital, Box 5339, London, ON N6A 5K8**Canada
JOURNAL: Immunology 91 (1):p135-144 1997
ISSN: 0019-2805
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The mechanisms contributing to the proliferation and differentiation of antigen-presenting cell (APC) precursors upon antigen stimulation or tissue injury are poorly understood. Herein, we report the induction of a population of dendritic cell-like cells (DLC) with potent antigen-presentation function from unfractionated spleen cells by means of repetitive allostimulation in long-term mixed leucocyte cultures (LT-MLC). Initially, only a few adherent DLC were observed. By 4-6 weeks, however, there were large numbers of DLC which survived persistently. Features of these DLC are closely related to dendritic cells (DC), including: (1) dendritic, veiled or spiny-processed morphology; (2) expression of a wide array of leucocyte surface markers including DC-associated or restricted antigens: 33D1, NLDC-145, CD11c (N418), heat-stable antigen (HSA), CD44, B7-1 and B7-2; (3) ability to migrate to draining lymph nodes and white pulp area of spleen; (4) expression of high level of major histocompatability complex (MHC) class II molecules and (5) more potent mixed leucocyte reaction (MLR)-stimulating capacity than peritoneal macrophages and APC-enriched spleen cells. DLC-stimulated MLR was inhibited by monoclonal **antibodies** (mAbs) to **B7**-

1, B7-2, intracellular adhesion molecule-1 (ICAM-1), vascular cell adhesion molecule-1 (VCAM-1), leucocyte-function associated antigen-I (LFA-1) or very-late activation antigen-4 (VLA-4) by 30-55%. When maintained for more than 2 months, the DLC did not lose their MLR-stimulating activity, but many surface markers were downregulated except for Mac-2 and VCAM- 1, which remained stable or were up-regulated, respectively. In short-term culture, the addition of granulocyte-macrophage colony-stimulating factor (GM-CSF) or interleukin (IL)-2 enhanced proliferation of DLC, while **tumour** necrosis factor-alpha (TNF-alpha) and IL-4 did not. IL-4 suppressed not only 'spontaneous', but also GM-CSF-enhanced proliferation, suggesting that cytokines play a differential role in DLC proliferation. These results confirm that professional A.PC can proliferate in response to repetitive antigen stimulation, and their proliferation is differentially regulated by cytokines. A comparison study of DLC with typical DC is being carried out in our laboratory.

5/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10897652 BIOSIS NO.: 199799518797
Expression of B7-1 and B7-2 costimulatory molecules by human gastric epithelial cells: Potential role in CD4+ T cell activation during Helicobacter pylori infection.
AUTHOR: Ye Gang; Barrera Carlos; Fan Xuejun; Gourley William K; Crowe Sheila E; Ernst Peter B; Reyes Victor E(a)
AUTHOR ADDRESS: (a)Univ. Texas Med. Branch, Child. Hosp., C-66, 301 University Blvd., Galveston, TX 77555-0366**USA
JOURNAL: Journal of Clinical Investigation 99 (7):p1628-1636 1997
ISSN: 0021-9738
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Human gastric mucosal epithelial cells display class II MHC, the expression of which is increased during Helicobacter pylori infection. These observations suggest that the gastric epithelium may participate as antigen-presenting cells (APC) during local immune responses. The increase in class II MHC expression occurs in parallel with an elevation in gastric CD4+ T cell numbers within and adjacent to the epithelium. Since the expression of either B7-1 (CD80) or B7-2 (CD86) on APC is required for the activation of T cells, it was important to establish human gastric epithelial cells expressed those surface ligands. The expression of B7-1 and B7-2 was detected on human gastric epithelial cell lines and freshly isolated epithelial cells from gastric biopsies with specific **antibodies**. **B7-2** expression was higher than B7-1 at both protein and transcript levels and was increased after crosslinking class II MHC molecules on IFN-gamma-treated epithelial cells and in cells pretreated with the combination of IFN-gamma and H. pylori. Similarly, B7-2 expression was higher on gastric epithelial cells from H. pylori-infected tissues compared with those from uninfected specimens. To determine the function of these molecules on gastric epithelial cells, **antibodies** to **B7-1** and **B7-2** were shown to reduce the ability of the cells to stimulate alloreactive CD4+ T cells. These observations are the first to demonstrate that B7-1 and B7-2 are expressed on mucosal epithelial cells in situ. Thus, the expression of B7-1 and B7-2 by epithelial cells may allow them to act as APC in regulating local responses such as those that occur during infection with H. pylori.

5/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10420868 BIOSIS NO.: 199699042013
Inflammatory cells infiltrating human colorectal carcinomas express HLA class II but not B7-1 and B7-2 costimulatory molecules of the T-cell activation.
AUTHOR: Chaux Pascal; Moutet Monique; Faivre Jean; Martin Francois; Martin Monique(a)
AUTHOR ADDRESS: (a)Dep. Biol. Therapy Cancer, Fac. de Medecine, 7 Bd. Jeanne d'Arc, 21033 Dijon**France
JOURNAL: Laboratory Investigation 74 (5):p975-983 1996
ISSN: 0023-6837
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Colon **cancer** cell sex press potentially immunogenic proteins but are not rejected by the immune system. To induce an effective immune response, antigenic peptides have to be presented to T lymphocytes by professional antigen-presenting cells in association with HLA class II molecules. Antigen-presenting cells also have to express B7 family molecules, B7-1 and B7-2, which deliver the costimulatory signals that are required to prevent T cell anergy. We studied B7-1 and B7-2 expression by the antigen-presenting cells that infiltrate colorectal **cancer** stroma. In 25 samples of colorectal carcinomas, a panel of monoclonal antibodies was used to label macrophages, dendritic cells, and T lymphocytes that infiltrate the **tumor** stroma and the morphologically normal distant mucosa. The expression of HLA class II and B7 molecules involved in T-cell activation was studied using specific monoclonal antibodies. Biopsy pieces from two patients with active Crohn's disease were used as controls. All of the samples were heavily infiltrated by macrophages and/or dendritic cells that strongly expressed HLA class II molecules. In contrast, **antibodies** to **B7-1** and/or **B7-2** stained no cells in 16 of the 25 samples of colorectal **tumors** and less than 1% of the inflammatory cells that infiltrated **tumor** stroma of the other nine **tumor** samples. B7 molecules were also poorly expressed by rare cells in the lamina propria of the morphologically normal colorectal mucosa. In contrast, many inflammatory cells that infiltrated the two Crohn's disease samples strongly expressed B7-1 and B7-2, especially in the granulomas, We conclude that most HLA class II inflammatory cells that infiltrate colorectal **cancers** do not express the B7-1 and B7-2 costimulatory molecules. This defect may contribute to the failure of the immune system to recognize **tumor** cells as antigenic.

5/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10225261 BIOSIS NO.: 199698680179
Comparison of CD28-B7.1 and B7.2 functional interaction in resting human T cells: Phosphatidylinositol 3-kinase association to CD28 and cytokine production.
AUTHOR: Ghiotto-Ragueneau Marguerite; Battifora Michela; Truneh Alemsedeg; Waterfield Michael D; Olive Daniel(a)
AUTHOR ADDRESS: (a)INSERM U119, 27 Bd Lei Roure, F-13009 Marseille**France
JOURNAL: European Journal of Immunology 26 (1):p34-41 1996
ISSN: 0014-2980
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: CD28 is a 44-kDa homodimer present on T cells providing an

important costimulatory signal for T cell proliferation, cytokine production and cytokine receptor expression. CD28 activation is mediated by interaction with its counter-receptors, B7.1/CD80 and B7.2/B70/CD86. The biochemical basis of these costimulatory signals are still poorly understood, particularly in resting T cells. However, various biochemical pathways such as tyrosine phosphorylation, phospholipase C, sphingomyelinase and phosphatidylinositol 3-kinase (PI3-K) activation have been reported to play a role in CD28 signaling in **tumor** T cell lines and CD28-transfected cells or pre-activated T cells. In addition, recent reports propose that CD28-B7.1 and B7.2 interaction could be involved in the production of Th1 and Th2 cytokines, respectively, but the putative biochemical basis for these different functions is still unknown. We have analyzed the functional and molecular consequences of CD28 activation by B7.1 and B7.2 in human resting T cells. We demonstrate in this report that both CD28-B7.1 and CD28-B7.2 interactions induce the association of PI3-K to CD28 in the CD4 subpopulation, whereas it was barely detectable in DD8 cells. This association involves the binding of the src homology domain 2 (SH2) of p85 to tyrosinephosphorylated CD28 and does not require pre-activation by CD3-T cell receptor. Worthmannin, a specific inhibitor of PI3-K enzymatic activity within the nanomolar range also inhibits the interleukin-2 production induced by coStimulation mediated by either the B7.1- and **B7**.2-transfected cells or CD28 monoclonal **antibodies**. The only slight difference between **B7**.
1 and B7.2 costimulation is the IC-50 of wortmannin being 25 and 110 nM, respectively, which could suggest differences in their activation of the T cell PI3-K.

5/7/8 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

10967908 EMBASE No: 2001005827
Expression and function of the costimulatory molecules B7-1 (CD80) and B7-2 (CD86) in an in vitro model of the human blood-brain barrier
Omari K.I.M.; Dorovini-Zis K.
K. Dorovini-Zis, Dept. of Pathology and Lab. Med., Vancouver General Hospital, University of British Columbia, 855 West 12th Avenue, Vancouver, BC V5Z 1M9 Canada
AUTHOR EMAIL: dorovini@interchange.ubc.ca
Journal of Neuroimmunology (J. NEUROIMMUNOL.) (Netherlands) 01 FEB 2001, 113/1 (129-141)
CODEN: JNRID ISSN: 0165-5728
PUBLISHER ITEM IDENTIFIER: S0165572800004355
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 72

The interaction of B7 molecules with their ligand provides important accessory signals for optimal T cell activation and proliferation. In this study the in vitro expression of B7-1 and B7-2 by human brain microvessel endothelial cells (HBMEC) was investigated by semiquantitative reverse transcriptase-polymerase chain reaction (RT-PCR) and immunocytochemistry. In addition, the contribution of B7 molecules to T cell proliferation on cerebral endothelial cells was studied by coincubating purified CD4+T cells with resting or cytokine activated HBMEC. Untreated cultures constitutively expressed B7-2 RNA and surface protein, but lacked B7-1 expression. Treatment with TNF-alpha and IFN-gamma upregulated B7-2 and induced de novo expression of **B7-1**. Monoclonal blocking **antibodies** to **B7-1** or **B7-2** and human CTLA-4Ig chimeric protein significantly reduced the ability of HBMEC to support alpha-CD3-induced proliferation of CD4+ T lymphocytes. Expression of B7 glycoproteins and the ability to provide secondary signals for T cell proliferation suggest a potential role of the human cerebral endothelium in T cell activation

during the early stages of central nervous system inflammation. (c) 2001 Elsevier Science B.V.

5/7/9 (Item 2 from file: 73)
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07738119 EMBASE No: 1999220681
Interaction between B.7 and CD28 costimulatory molecules is essential for the activation of effector function mediating spontaneous **tumour** regression
Rao K.L.; Varalakshmi Ch.; Kumari A.L.; Khar A.
A. Khar, Ctr. for Cellular/Molecular Biology, Uppal Road, Hyderabad 500 007 India
Scandinavian Journal of Immunology (SCAND. J. IMMUNOL.) (United Kingdom) 1999, 49/6 (633-640)
CODEN: SJIMA ISSN: 0300-9475
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 47

The spontaneous regression of a rat histiocytoma, AK-5, is mediated by activated natural killer cells through antibody-dependent cellular cytotoxicity. In addition to the Fc-FCR interaction between the target and the effector cells demonstrated previously, we show the participation of costimulatory molecules B7 and CD28 in the efficient killing of the **tumour** cell. Blockade of the costimulatory interaction in vivo using anti-CD28 led to increased **tumour** growth and a suppressed cytokine response. Anti-CD28 antibody administration in vivo also diminished the cytotoxic potential of NK cells against AK-5 cells in vitro. Our studies also demonstrate the expression of B7.1 and B7.2 antigen on AK-5 **tumour** cells. The cytotoxic activity of natural killer cells was significantly inhibited when the effector/target cells were cultured in the presence of **antibodies** raised against **B7.1**, **B7.2** and CD28. Administration of anti-CD28 in vivo also affected the efficiency of the formation of effector/target conjugates in vitro. Similarly, anti-CD28 injections affected expression of the adhesion molecules LFA 1 and ICAM 1 by splenocytes. Administration of anti-**B7.1** and **B7.2 antibodies** in AK-5 **tumour**-bearing animals showed a differential response. The cytotoxicity of natural killer cells was significantly inhibited after anti-B7.2 administration, suggesting the preferential participation of B7.2 molecules in vivo. These observations suggest an important role for B7-CD28 interaction in AK-5 **tumour** regression.

5/7/10 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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135240937 CA: 135(17)240937k PATENT
Use of a combination of agents that modulate B7 activity in inhibiting intestinal allograft rejection
INVENTOR(AUTHOR): Collins, Mary; Newell, Kenneth
LOCATION: USA
ASSIGNEE: Genetics Institute, Inc.
PATENT: PCT International ; WO 200168132 A1 DATE: 20010920
APPLICATION: WO 2001US8015 (20010313) *US PV189165 (20000314)
PAGES: 56 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A; A61K-031/445B; A61P-037/06B; A61K-039/395B; A61K-031/445B
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV;

MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG ; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA215010 Immunochemistry
CA201XXX Pharmacology
IDENTIFIERS: intestinal allograft survival B7 antibody rapamycin
DESCRIPTORS:
Transplant and Transplantation...
allotransplant, small intestine; use of antibodies to B7-1 and B7-2 and a rapamycin compd. in inhibiting intestinal allograft rejection
Chemokine receptors...
.beta. chemokine receptor CCR5; inhibiting cytokine prodn. and the CD28/B7 pathway by anti-B7 antibodies in relation to inhibiting intestinal allograft rejection
Interferons...
.gamma.; inhibiting cytokine prodn. and the CD28/B7 pathway by anti-B7 antibodies in relation to inhibiting intestinal allograft rejection
CD28(antigen)... Interleukin 12... Interleukin 2... RANTES(chemokine)...
Tumor necrosis factors...
inhibiting cytokine prodn. and the CD28/B7 pathway by anti-B7 antibodies in relation to inhibiting intestinal allograft rejection
Chemokines...
macrophage inflammatory protein 1; inhibiting cytokine prodn. and the CD28/B7 pathway by anti-B7 antibodies in relation to inhibiting intestinal allograft rejection
Antibodies...
monoclonal; use of antibodies to B7-1 and B7-2 and a rapamycin compd. in inhibiting intestinal allograft rejection
Intestine...
small, allotransplant; use of antibodies to B7-1 and B7-2 and a rapamycin compd. in inhibiting intestinal allograft rejection
Antibodies... CD80(antigen)... CD86(antigen)... Immunosuppression...
Immunotherapy... Signal transduction,biological...
use of antibodies to B7-1 and B7-2 and a rapamycin compd. in inhibiting intestinal allograft rejection
CAS REGISTRY NUMBERS:
53123-88-9D derivs., use of antibodies to B7-1 and B7-2 and a rapamycin compd. in inhibiting intestinal allograft rejection
53123-88-9 use of antibodies to B7-1 and B7-2 and a rapamycin compd. in inhibiting intestinal allograft rejection

5/7/11 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

134265164 CA: 134(19)265164y PATENT
Human and mouse GL50 proteins, their sequences, recombinant production, and use in screening, and therapeutic methods
INVENTOR(AUTHOR): Ling, Vincent; Dunussi-Joannopolulos, Kyriaki
LOCATION: USA
ASSIGNEE: Genetics Institute, Inc.
PATENT: PCT International ; WO 200121796 A2 DATE: 20010329
APPLICATION: WO 2000US25892 (20000921) *US PV155043 (19990921)
PAGES: 194 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-015/12A; C07K-014/705B; C12N-015/11B; G01N-033/53B; G01N-033/68B; C07K-016/28B; A61K-038/17B; A61K-039/395B DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CR; CU; CZ; DE; DK; DM; DZ; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; UZ; VN; YU; ZA; ZW;

AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW ; MZ; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

SECTION:

CA215010 Immunochemistry
CA201XXX Pharmacology
CA203XXX Biochemical Genetics
CA214XXX Mammalian Pathological Biochemistry

IDENTIFIERS: cDNA sequence human mouse GL50 antigen mol cloning, immune response modulation GL50 antigen T cell costimulation, antitumor agent GL50 antigen mouse

DESCRIPTORS:

Antibodies...
anti-B7-1 or anti-B7-2; modulating immune response by administering anti-B7-1 or anti-B7-2 antibodies

Antibodies...
anti-GL50; modulating immune response by administering a GL50 modulating agent or by administering anti-GL50 antibody

T cell(lymphocyte)...
co-stimulation; modulating T cell costimulation by contacting activated T cell with GL50 protein

Animal cell line...
COS; human and mouse GL50 proteins, their sequences, recombinant prodn., and use in screening, and therapeutic methods

Protein motifs...
extracelluar domain; reducing proliferation of tumor cell by allowing immune cell to contact an activated form (contg. extracellular domain) of GL50 protein

Fusion proteins(chimeric proteins)...
fusion proteins comprising human and mouse GL50 proteins linked to IgGa

Antigens...
GL50; human and mouse GL50 proteins, their sequences, recombinant prodn., and use in screening, and therapeutic methods

Immunoglobulins...
G2a, fused to mouse GL50-1 and human GL50; fusion proteins comprising human and mouse GL50 proteins linked to IgGa

cDNA sequences... Mouse...
human and mouse cDNA mols. encoding GL50 proteins, their sequences and use in recombinant prodn. of GL50

Molecular cloning... Plasmid vectors... Protein sequences...
human and mouse GL50 proteins, their sequences, recombinant prodn., and use in screening, and therapeutic methods

Drug screening...
method for screening for a compd. that modulates GL50 mediated activation of immune cells

Signal transduction,biological...
method for screening for a compd. that modulates signal transduction in immune cell

Immunity...
modulating immune response by administering a GL50 modulating agent or by administering anti-GL50 antibody

Antisense oligonucleotides...
nucleic acid mol. comprising a sequence complementary to human and mouse GL50 cDNAs

Antitumor agents...
reducing proliferation of tumor cell by allowing immune cell to contact an activated form of GL50 protein

Neoplasm...
reducing proliferation of tumor cell by allowing immune cell to contact GL50 protein

CAS REGISTRY NUMBERS:

261150-60-1P 264902-87-6P 331694-58-7P amino acid sequence; human and mouse GL50 proteins, their sequences, recombinant prodn., and use in

screening, and therapeutic methods
331615-97-5 331615-98-6 331615-99-7 nucleotide sequence; human and mouse cDNA mols. encoding GL50 proteins, their sequences and use in recombinant prodn. of GL50
245735-23-3 247032-91-3 294216-44-7 294216-45-8 332002-46-7 332002-47-8 332002-48-9 332002-49-0 332002-50-3 332002-51-4 332002-52-5 332002-53-6 332002-54-7 332002-55-8 332002-57-0 332087-36-2 unclaimed nucleotide sequence; human and mouse GL50 proteins, their sequences, recombinant prodn., and use in screening, and therapeutic methods
332002-56-9 332087-38-4 unclaimed protein sequence; human and mouse GL50 proteins, their sequences, recombinant prodn., and use in screening, and therapeutic methods
332087-35-1 332087-37-3 332087-39-5 332087-40-8 unclaimed sequence; human and mouse GL50 proteins, their sequences, recombinant prodn., and use in screening, and therapeutic methods
331969-80-3 Unclaimed; human and mouse GL50 proteins, their sequences, recombinant prodn., and use in screening, and therapeutic methods
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